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Inhibition of return and nonspecific preparation: Separable inhibitory control mechanisms in space and time

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I examined the relation between two inhibitory processes operating on spatial and temporal representations. In two experiments, participants had to detect a peripheral target that was presented after a variable interval following the onset of an uninformative peripheral cue. For the shortest cue–target interval, target detection was faster at the cued than at the uncued location, but this effect was reversed for the longer cue–target intervals. This finding has been taken to reflect a buildup of space-related inhibition over time, known as *inhibition of return*. Also, target detection was slower when the cue–target interval of the preceding trial was longer than that of the current trial than when this was not so. This sequential effect has been taken to reflect an intertrial carryover of time-related inhibition. Crucially, the spatial and temporal effects were additive in both experiments, suggesting a modular organization of the underlying inhibitory processes.

With the renewed interest in executive processes, inhibitory control has become a popular theoretical construct in experimental psychology. It has been invoked to explain the ability of people to make correct decisions when inconsistent elements in the spatiotemporal environment compete for another, incorrect decision. The mental system is apparently capable of suppressing the undesired outcome or the processing that gives rise to it, which naturally leads to the postulation of inhibitory control mechanisms. Thus, the past 2 decades have seen a proliferation of proposed inhibitory control mechanisms, involved in a variety of cognitive domains, yet with the same ultimate aim of preventing undesired outcomes from showing up in behavior (for reviews, see Arbuthnott, 1995; Band & van Boxtel, 1999).

In this study, I examined the relation between two purported inhibitory mechanisms, one acting on representations of space and another acting on representations of time. I used a variant of Posner and Cohen's (1984) spatial-cuing paradigm, because of the potential it has to reveal possible interactions between these inhibitory mechanisms. The role of inhibition in space and time has been reflected in experimental phenomena described in different literatures. In what follows, I will introduce these phenomena and their underlying inhibitory mechanisms in turn, followed by a presentation of the experimental plan of this study.

Inhibition in Space

In the spatial-cuing paradigm of present interest, a trial starts with a brief presentation of a peripheral cue, marking a potential target location on the left or right of central fixation. The participant is instructed to abstain from responding to the cue and to maintain central fixation. After a variable stimulus onset asynchrony (SOA), the cue is followed by a peripheral target that occurs, with equal probability, either at the cued location or at the uncued location on the opposite side of central fixation. The participant is instructed to make a fast manual (or saccadic) response with respect to a predefined aspect of the target.

In this version of the spatial-cuing paradigm, the cue is task irrelevant and provides no information about the impending target location. Nevertheless, a characteristic pattern of interference is invariably reported, showing that, as SOA increases, an initial facilitation in the reaction time (RT) for the target at the cued location turns into inhibition. Specifically, for brief SOAs (typically, below 150 msec), RT is usually shorter when the target appears at the cued location than when it appears at the uncued location (e.g., Klein, 2000; Maylor, 1985; Posner & Cohen, 1984; Pratt, Hillis, & Gold, 2001). This effect is reversed for longer SOAs (typically, above 300 msec) and stabilizes a few hundred milliseconds later. Because for long SOAs, RTs are longer for targets appearing at the cued location than for those at the uncued location, it has been suggested that observers are inhibited from returning to an already attended location—hence, the term *inhibition of return* (IOR; Posner, Rafal, Choate, & Vaughan, 1985; for recent reviews, see Klein, 2000; Taylor & Klein, 1998).

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An element common to many interpretations of the interaction between cuing and SOA is that a location-specific inhibitory process is elicited by the cue that comes to dominate a more transient cue-elicited facilitatory process. The initial idea was that the inhibitory process is contingent on the facilitatory process (e.g., Maylor, 1985), but recent evidence suggests that these processes develop in parallel, whereby the fast but transient facilitation process initially masks the more slowly developing inhibition process (Danziger & Kingstone, 1999). In this article, I will not go into this discussion and will focus on the inhibitory process. The central assumption is that the cue elicits an inhibitory process that develops during the cue–target interval and comes to interfere with the subsequent target-related process, provided that the target appears at the cued location.

Although the origin of inhibition and the locus of its influence in the processing chain for the target has been controversial since the first reports on IOR (e.g., Klein, 2000), the role of inhibition itself has gone relatively unchallenged (see Pratt, Spalek, & Bradshaw, 1999, for a notable exception and Snyder, Schmidt, & Kingstone, 2001, for a rebuttal). In fact, inhibitory influences have been traced down to the neural level in studies in which single-cell recordings in the monkey have been used. These studies have shown reduced neural activity when the target has appeared at the cued, as compared with the uncued, location in brain areas that have been associated with IOR, such as the superior colliculus (Dorris, Klein, Everling, & Munoz, 2002) and the lateral intraparietal sulcus (Robinson, Bowman, & Kertzman, 1995). These and other findings are consistent with the view that inhibition shapes a participant's representation of space or of objects as they move about in space (Müller & von Mühlenen, 2000; Tipper, Weaver, Jerreat, & Burak, 1994). The functional relevance of this mechanism is supposed to be to prevent the orienting system from revisiting locations or objects that were visited shortly before, thus enhancing efficiency of the search for a target in the environment (Klein, 1988, 2000).

Inhibition in Time

The spatial-cuing paradigm has attracted many researchers interested in how inhibitory processes come to shape a participant's representation of space. What has so far been ignored, though, is that, in this paradigm, inhibitory processes are involved in shaping not only representations of space, but also those of time.

The role of inhibition in shaping the representation of time has been derived from recent insights into the mechanism underlying *nonspecific preparation.* Nonspecific preparation refers to the fluctuations over time in the general readiness to respond to an anticipated target stimulus after the occurrence of a time marker, such as a warning stimulus or a cue. These fluctuations are particularly robust in experimental designs in which two or more clearly distinct levels of SOA are varied randomly and equiprobably across trials. The classical finding in such designs has been that RT decreases as SOA increases according to a negatively accelerating function (e.g., Woodrow, 1914; Wundt, 1887; see Niemi & Näätänen, 1981, for a review). However, this classical main effect has limited significance in itself, because it is strongly modified by sequential influences of SOA. In particular, RT on a given trial n is longer when the SOA of that trial (SOA_n) is shorter than the SOA of the preceding trial (SOA_{n-1}), as compared with when SOA_n is as long as or longer than SOA_{n-1} (see, e.g., Baumeister & Joubert, 1969; Drazin, 1961; Los, Knol, & Boers, 2001; Los & van den Heuvel, 2001; Woodrow, 1914). Note that this sequential effect is asymmetric in that the effect of SOA_{n-1} decreases as SOA_n increases. In fact, this asymmetry is so strong that it deprives the classical main effect of SOA_n of any independent significance (e.g., Los & van den Heuvel, 2001). Thus, the sequential effect of SOA fulfills a key role in the understanding of the process of nonspecific preparation.

Traditional explanations of this sequential effect have all conceived of nonspecific preparation as an activating process driven by expectancies as to when the target is likely to occur (Niemi & Näätänen, 1981; Requin, Brener, & Ring, 1991). Apart from having particular theoretical shortcomings (see Los & van den Heuvel, 2001, for a discussion), these accounts have failed to incorporate the robust finding from psychophysiology that inhibition plays an important role during the cue–target interval. For instance, during this interval, physiological indices, such as the heart rate (e.g., Bohlin & Kjellberg, 1979; Jennings, van der Molen, & Steinhauer, 1998), the pupil diameter (e.g., Jennings et al., 1998), and the Achilles tendon reflex (e.g., Brunia & Boelhouwer, 1988), show inhibitory effects relative to their appropriate baselines. A common interpretation of these effects has been that an inhibitory mechanism prevents premature responding during the cue–target interval (Brunia, 1993).

Recently, Los and colleagues (Los, 1996; Los et al., 2001; Los & van den Heuvel, 2001) have assigned a key role to this inhibition mechanism in their trace-conditioning account of nonspecific preparation. According to the simplest version of this account, participants rapidly learn (during a few practice trials) the moments at which the target may occur after the onset (or offset) of the cue, which are referred to as *critical moments.* As a result of this learning, the cue becomes a conditioned stimulus, which entails a conditioned tendency to respond at a critical moment as it is approached during the cue–target interval (e.g., Gallistel & Gibbon, 2000; Machado, 1997). To prevent premature responding, an inhibitory mechanism serves to suppress the conditioned response as the critical moment is bypassed during the cue–target interval, resulting in a reduced (or extinguished) state of conditioning being associated with that critical moment. Inhibition stops and reinforcement takes over at the moment of target presentation, causing the state of conditioning associated with the critical moment of target occurrence to increase. Finally, the states of conditioning associated with critical moments beyond the moment of target occurrence are subject neither to extinction nor to reinforcement.

The learning rules implied by this account are illustrated in Figure 1. Figure 1A shows a hypothetical equal state of conditioning associated with each of four critical moments at the start of a given trial. When the target happens to occur at the third critical moment of that trial, the inhibitory mechanism suppresses the conditioned response associated with the first two critical moments as they are bypassed during the cue–target interval (Figure 1B). This results in extinction of the states of conditioning associated with these critical moments (Figure 1C). Furthermore, the state of conditioning associated with the critical moment of target occurrence is increased toward a certain upper asymptotic value, whereas the states of conditioning associated with critical moments beyond the moment of target occurrence (the fourth critical moment in the example) remain unchanged. These dynamics result in an overall state of conditioning, shown in Figure 1D. This state of conditioning is preserved for the next trial.

On the assumption that RT is inversely related to the state of conditioning associated with the critical moment of target occurrence, the asymmetric sequential effect of SOA can be readily explained. The inhibitory mechanism reduces only the state of conditioning associated with critical moments that are bypassed during the cue–target interval, and not the state of conditioning associated with later critical moments. Therefore, a relatively long RT should be observed only when SOA_n is shorter than SOA_{n-1}, consistent with the asymmetric sequential effect of SOA.

In a recent study, Los (2004) obtained behavioral evidence that response inhibition during the cue–target interval lies at the origin of the sequential effects of SOA. In this study, regular go trials were intermixed with *forewarned no-go trials*, on which participants were told at the start of the trial to relax and not to respond to the impending target stimulus. On the go trials after these forewarned no-go trials, responding was generally fast and nearly independent of the duration of the cue–target interval that had been used on the preceding no-go trial. This finding suggests that in the absence of any intention to respond to the target (on forewarned no-go trials), there is no response tendency as a critical moment is bypassed and, therefore, no need for inhibition. This in turn gives rise to fast responding on the subsequent go trial, even when the moment of target occurrence was bypassed on the preceding no-go trial.

To summarize, the conditioning view proposed by Los and colleagues (Los, 1996; Los et al., 2001; Los & van den Heuvel, 2001) assigns a key role to response inhibition in shaping the representation of time. Although this role is relatively unexplored, it is consistent with physiological evidence showing response-related inhibition in anticipation of a target, whereas preliminary data (Los, 2004) clearly bear out the relationship between response inhibition and sequential effects of SOA.

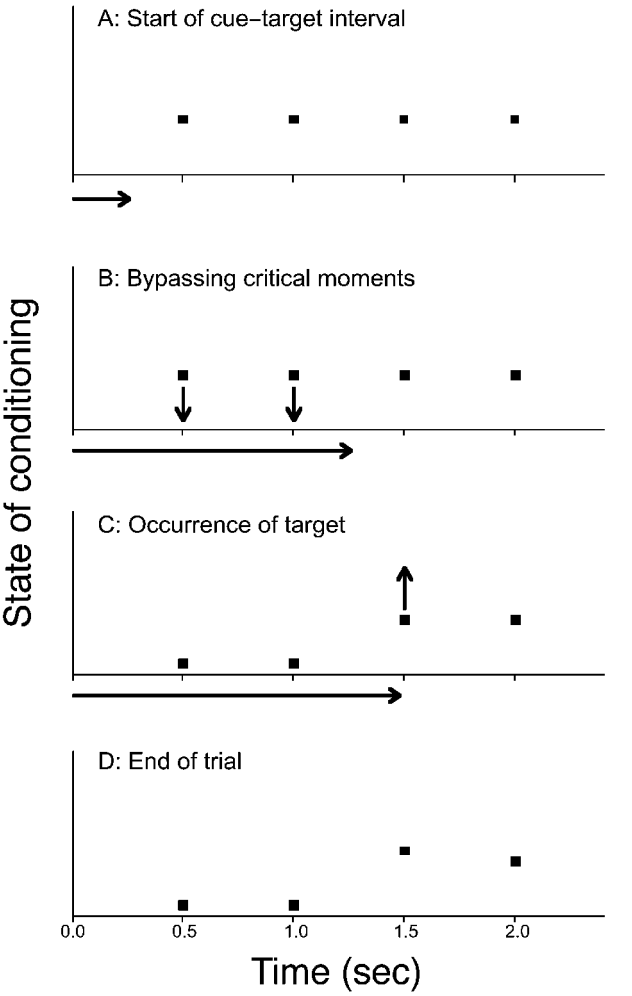

Figure 1. The conditioning theory of nonspecific preparation. Time is represented in terms of a state of conditioning associated with each critical moment (i.e., potential moment of target occurrence). (A) Hypothetical equal state of conditioning associated with each of four critical moments at the start of a given trial. (B) Upon bypassing a critical moment during the cue–target interval, the associated state of conditioning is subject to extinction. (C) The state of conditioning associated with the critical moment of target occurrence is reinforced. (D) The resulting state of conditioning after the trial, which is preserved for the next trial.

Inhibition in Space and Time

Combining the perspectives on IOR and nonspecific preparation, we see two similar inhibitory mechanisms at work during the cue–target interval, one shaping the representation of space and another shaping the representation of time. When the cue appears in the periphery, it tells the mental system *not here*, which is subsequently represented as *not there* relative to the point of fixation. During the same cue–target interval, a temporal mechanism tells the mental system *not now* as a critical moment is bypassed, which is subsequently represented as *not then* relative to the moment of the offset (or perhaps the onset) of the cue. In this study, I examined to what extent these mechanisms operate independently by testing the three-way interaction among cuing, SOA_n, and SOA_{n-1}. This interaction should be significant if the mechanisms underlying IOR and nonspecific prepara-

tion operate interdependently; it should be nonsignificant if these mechanisms operate independently.

To be more specific, I will outline two possible ways in which an interdependence between the mechanisms operating in space and time may give rise to a three-way interaction among SOA_n, SOA_{n-1}, and cuing. A first possible mechanism relies on the assumption that inhibition operates on representations in space-time, and not on separate representations in space and time. According to this view, the expression of IOR may be as much dependent on the temporal correspondence between SOA_n and SOA_{n-1} as it is on the spatial correspondence between the cue and the target (e.g., Maylor & Hockey, 1985). Consequently, the inhibitory landscape in space-time may reach its summit when the target occurs at the cued location and at the same critical moment as that at which the target occurred on the preceding trial. This should give rise to a three-way interaction among SOA_n, SOA_{n-1}, and cuing. A second possibility is that inhibition of temporal and spatial representations emanates from a common source, so that stronger inhibition in one domain implies reduced inhibition in the other domain. Regarding nonspecific preparation, it is plausible to assume that the demand for inhibition in approaching a critical moment is stronger to the degree to which the state of conditioning associated with that critical moment is higher. That is, there is a greater demand for inhibition upon approaching a critical moment when on the preceding trial the target occurred at or before that critical moment than when it occurred at a later moment. Therefore, if the temporal and spatial mechanisms draw from a common source of inhibition, IOR for a given SOA should be larger when a longer SOA occurred on the preceding trial, as compared with when an equally long or shorter SOA occurred on the preceding trial, again resulting in a three-way interaction.

When neither of these possibilities turns out to be correct—that is, when separate sources of inhibition subserve the suppression of separate representations in space and time—the temporal and spatial mechanisms will operate as separate modules, rendering a significant three-way interaction among SOA_n, SOA_{n-1}, and cuing unlikely. Indeed, a nonsignificant three-way interaction would follow compellingly from an architecture in which modules operate without temporal overlap, in separate stages (Sternberg, 1969, 2001), but it may also be obtained when modules operate under (partial) temporal overlap (McClelland, 1979; Miller, van der Ham, & Sanders, 1995). Conversely, under both these architectures, a significant three-way interaction is a highly likely outcome whenever three factors affect at least one common module. Thus, by applying the *modus tollens*, finding IOR to be independent of sequential effects of SOA would be strong evidence for a modular organization of a temporal and spatial inhibitory mechanism.

To summarize, the test of the three-way interaction among cuing, SOA_n, and SOA_{n-1} may provide a theoretically important insight into the organization of inhibitory control processes. On the one hand, a significant three-way interaction would indicate that the inhibitory phenomena in space and time are different manifestations of a single unitary system. On the other hand, a nonsignificant three-way interaction would indicate a diversity of inhibitory control mechanisms operating in separate domains of cognitive functioning. Apart from these theoretical considerations, a more practical reason for testing the three-way interaction is that effects of nonspecific preparation are part and parcel of any spatial-cuing paradigm—at least insofar as more than one critical moment is included in the experimental design. So far, researchers have interpreted only the spatial effects of this paradigm. For future explorations, it seems useful to know whether possible interactions with the temporal effects of this paradigm should be considered.

The task used in this study has been a very common one in research into IOR (cf. Klein, 2000). It requires participants to detect a target that can occur at one of two equiprobable locations and at one of three equiprobably critical moments after the onset of an uninformative peripheral cue. To make sure that the results were not dependent on the idiosyncrasies of a specific SOA range, I will report on two experiments. In Experiment 1, SOA was varied at 100, 200, and 500 msec; in Experiment 2, SOA was varied at 100, 500, and 900 msec. In both experiments, 20% of the trials were catch trials on which no target occurred. Apart from preventing anticipatory behavior when the target was presented at the latest critical moment, these catch trials enabled testing of the novel prediction, from the conditioning view of nonspecific preparation, that responding at the latest critical moment will be relatively slow after a catch trial, because, on catch trials, the latest critical moment is bypassed while the participant is still in anticipation of a possible target. This should result in inhibition of the state of conditioning associated with the latest critical moment and, thus, in a relatively long RT when, on the next trial, the target happens to occur at the latest critical moment.

EXPERIMENT 1

Method

Participants. Twelve students with normal or corrected-to-normal vision participated in a single 1-h session for payment of 7 Euro.

Materials. The laboratory consisted of five identical, dimly lit, air-conditioned cubicles. Each cubicle was equipped with a personal computer connected to a 17-in. color monitor and a standard QWERTY keyboard. The participant sat in one of the cubicles with his or her head supported by a chinrest at a distance of 85 cm from the monitor and with the index finger of his or her preferred hand resting on the space bar of the keyboard. The ERTS software package (Beringer, 1992) was used to program and run the experiment and to register RTs. Visual stimuli were all white 12-cd/m^2 figures presented on the dark 0-cd/m^2 computer screen. The background display consisted of a central plus sign, flanked on both sides by the contours of a white square. The horizontal and vertical bars of the plus sign measured 2.5 × 13 mm. The squares measured 17 mm, or 1.15°, of visual angle, and the width of their contours was 1 mm.

The distance of each square to the plus sign was, center to center, 10.5 cm, or 7° of visual angle.

Task. Figure 2 shows the sequence of events in an experimental trial. Each trial started with a 1,500-msec presentation of the background display. The participants were instructed to fixate the plus sign throughout the trial. Next, the cue was presented for 50 msec, by outwardly doubling the thickness of the contours of one of the squares. This was followed again by the presentation of the background display. On target trials, one of the squares was filled white after a variable SOA of 100, 200, or 500 msec following the onset of the cue. The filled-in square was the target stimulus, and the participants were instructed to press the space bar as soon as they detected it. The screen turned blank after the response or after a maximum interval of 1,000 msec had expired since the onset of the target, whichever occurred earlier. On catch trials, no target stimulus occurred, and the participants were instructed not to respond. On these trials, the screen turned blank after an equiprobable interval of 1,100, 1,200, or 1,500 msec. Failures to respond within a 150- to 1,000-msec interval after target onset or responses given on catch trials elicited a 50-msec, 400-Hz tone when the screen turned blank. Between subsequent trials, the screen remained blank for 1,000 msec.

Design and Procedure. The independent variables were cuing (cued or uncued) and SOA (100, 200, or 500 msec). These variables were varied randomly within subjects and within blocks of trials.

The participants received written task instructions, which emphasized speed and accuracy equally. Next, they completed 2 practice blocks of 32 trials each and 18 experimental blocks of 62 trials each. An experimental block started with 2 practice trials. The remaining 60 trials contained a random mixture of 12 catch trials (20%) and 48 target trials. The target trials consisted of four replications of each factorial combination of SOA (three levels), cue position (left or right), and target position (left or right). Note that this trial composition implies that the cue was uninformative regarding the location of the impending target.

After the completion of each block, the participants saw the mean RT and the number of errors for that block presented on the screen. After each practice block, these scores were inspected by the experimenter, who encouraged the participants to respond faster when mean RT was above 350 msec and more accurately when a response occurred on more than two catch trials. After each of the experimental blocks, the participants copied the RT and error scores on a sheet of paper, to help them maintain high performance throughout the experiment.

Results

Data from practice blocks and from the first two trials of each subsequent block were excluded from further analysis. In the analysis of RTs, all target trials were included that had RTs occurring within a 150- to 800-msec interval after target onset. Figure 3 shows mean RT as a function of cuing, SOA_n, and SOA_{n-1}. These data were subjected to a repeated measures univariate analysis of variance (ANOVA), with α set at .05. The reported values for MS_e and p correspond to the Huynh–Feldt correction for violations of the sphericity of the variance–covariance matrix (see, e.g., Stevens, 1992). Table 1 provides a summary of the ANOVA results. Of crucial importance is the finding that the three-way interaction among cuing, SOA_n, and SOA_{n-1} failed to reach significance ($p = .35$).

The interaction between cuing and SOA_n reflects the fact that an initial facilitation of processing at the cued location turned into IOR as the cue–target interval increased. In Figure 3, this interaction is shown by the fact that across subsequent panels, RT in the cued condition (averaged across SOA_{n-1}) is increasingly longer relative to RT in the uncued condition. Specifically, relative to RT in the uncued condition, RT in the cued condition was 10 msec shorter when SOA_n was 100 msec [$F(1,11) = 8.63, MS_e = 302.96, p < .05$], 5 msec longer when SOA_n was 200 msec [$F(1,11) = 1.47$, $MS_e = 410.47$, $p = .25$, n.s.], and 34 msec longer when SOA_n was 500 msec [$F(1,11) = 86.71$, $MS_e = 312.56$, $p < .001$]. The interaction between SOA_n and SOA_{n-1} by and large reflects the fact that responding is slower to the extent that SOA_n is shorter than SOA_{n-1}. This pattern also

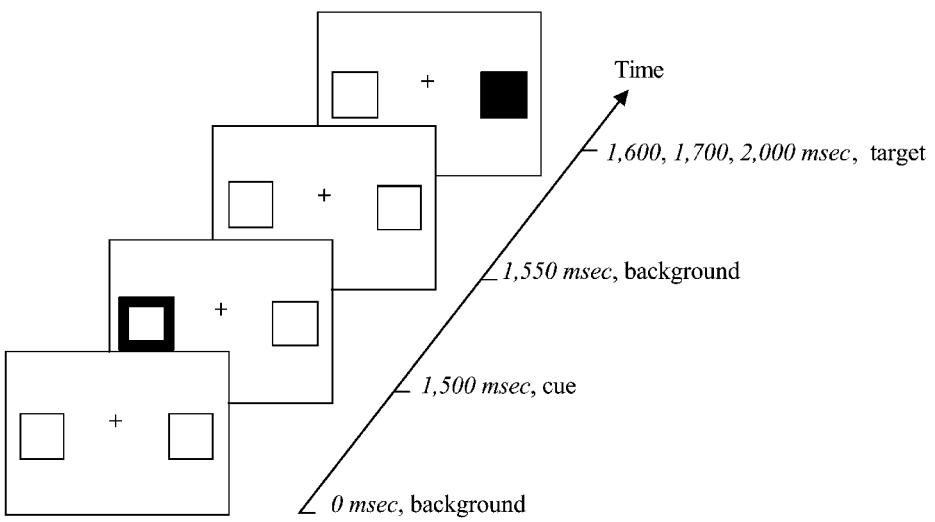

Figure 2. Time course of events in a target trial, shown with reversed figure–ground colors. In this example, the target appears at the uncued location.

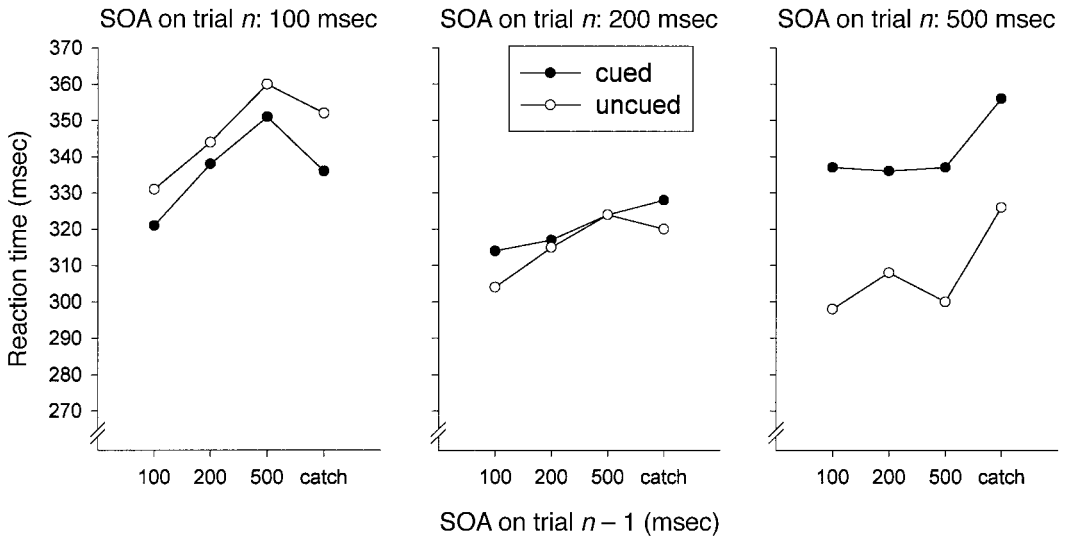

Figure 3. Mean reaction time in Experiment 1 as a function of cuing (cued or uncued), stimulus onset asynchrony (SOA) on trial *n*, and SOA on trial *n*−1.

holds for the longest SOA_n, where responding after a preceding catch trial was significantly slower than that after any other SOA_{n-1} [$F(1,11) > 36, p < .001$, for all paired comparisons]. A notable deviation from this pattern was observed for the shortest SOA_n, where responding after a preceding catch trial was faster than that after a preceding long SOA_{n-1} [$F(1,11) = 14.26, MS_e = 104.14, p < .01$].

Errors were classified as misses or false alarms. Misses were failures to respond within 800 msec after target onset. False alarms were responses on catch trials and responses earlier than 150 msec after target onset. Misses occurred very rarely, on 0.14% of the target trials, and were therefore not further analyzed. False alarms occurred on 2.77% of all the trials. As a function of SOA_{n-1}, with levels of 100 msec, 200 msec, 500 msec, and catch trial, the percentages of false alarms were 4.37%, 3.09%, 1.56%, and 2.07%, respectively. The ANOVA of this data revealed that the effect of SOA_{n-1} was significant [$F(3,33) = 15.42, MS_e = 1.40, p < .001$]. Post hoc tests indicated that all paired comparisons of SOA_{n-1} were significant [minimal $F(1,11) = 10.14, MS_e = 1.38, p < .01$], except for the comparison between an SOA_{n-1} of 500 msec and a preceding catch trial [$F(1,11) = 1.54, MS_e = 1.03, p = .24$]. The response latency of the false alarms averaged 355 msec since the onset of the cue, with 97.04% of these responses occurring within an 150- to 500-msec interval.

Discussion

The results of Experiment 1 replicated and extended earlier findings. The interaction between SOA_n and cuing has been the standard finding in the literature on IOR (Klein, 2000; Posner & Cohen, 1984). This finding has generally been taken to reflect a location-specific build up of inhibition during the cue–target interval. The interaction between SOA_n and SOA_{n-1} has been the standard finding in the literature on nonspecific preparation (Drazin, 1961; Niemi & Näätänen, 1981; Woodrow, 1914). Recent evidence suggests that this finding reflects inhibition of a temporal representation, brought about by bypassing the moment of target presentation during the cue–target interval on trial $n-1$ (Los, 2004; Los et al., 2001; Los & van den Heuvel, 2001). The novel finding in Experiment 1 was the absence of a three-way interaction among SOA_n, SOA_{n-1}, and cuing. This finding suggests that the inhibitory mechanisms affecting representations in space and time operate independently. Before discussing this issue in detail, I will report on a second experiment, which served to replicate the findings of Experiment 1, using a different SOA range.

EXPERIMENT 2

Method

Except for a different SOA range, the method in Experiment 2 was the same as that in Experiment 1. The SOAs in Experiment 2 were equiprobably 100, 500, and 900 msec. As in Experiment 1, 20% of the trials were catch trials. Twelve students with normal or corrected-to-normal vision participated in a single 1-h session for payment of 7 Euro. None of these students had participated in Experiment 1.

Results

The data were analyzed in the same way as in Experiment 1. Figure 4 shows mean RT as a function of cuing, SOA_n, and SOA_{n-1}. Table 1 presents a summary of the ANOVA results with Huynh–Feldt corrected values for MS_e and p. As Figure 4 and Table 1 show, there was strong agreement between the results of Experiment 1 and Experiment 2. Notably, in Experiment 2, there were highly significant interactions between cuing and SOA_n and between SOA_n and SOA_{n-1}, whereas the three-way

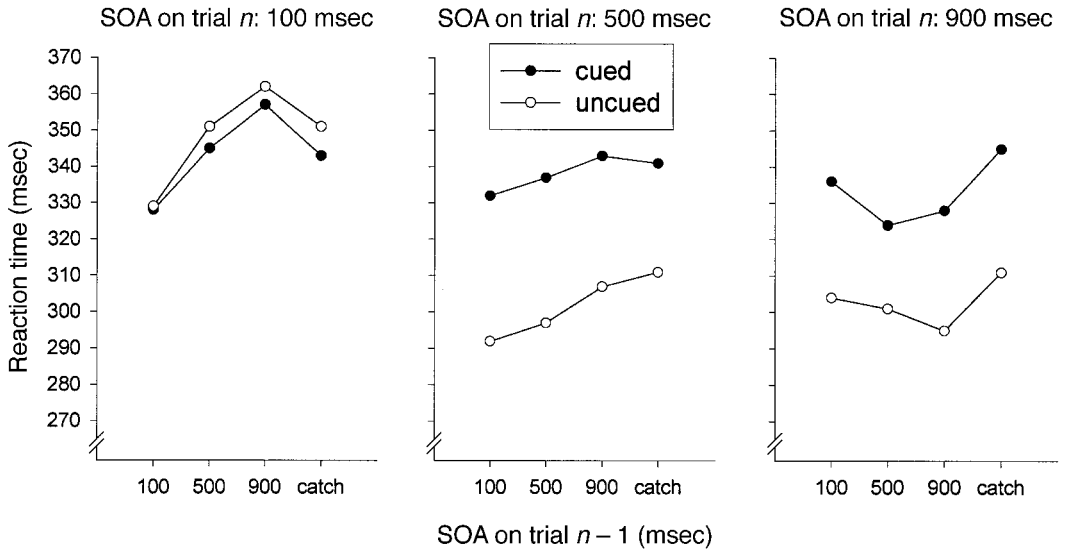

Figure 4. Mean reaction time in Experiment 2 as a function of cuing (cued or uncued), stimulus onset asynchrony (SOA) on trial *n*, and SOA on trial *n*−1.

interaction among cuing, SOA_n, and SOA_{n-1} was far from significant ($p = .45$).

The interaction between cuing and SOA_n reflects that relative to RT in the uncued condition, RT in the cued condition was 5 msec shorter when SOA_n was 100 msec [$F(1,11) = 1.94, MS_e = 324.99, p = .19$, n.s.], 36 msec longer when SOA_n was 500 msec [$F(1,11) = 27.68$, $MS_e = 1{,}157.72, p < .001$], and 30 msec longer when SOA_n was 900 msec [$F(1,11) = 27.05$, $MS_e = 796.38$, $p < .001$]. The interaction between SOA_n and SOA_{n-1} again reflects the fact that, for a given SOA_n, responding is slower to the extent that the critical moment was bypassed during the cue–target interval on the preceding trial. The details of this interaction were also in close agreement with those in Experiment 1. In particular, for the longest SOA_n, responding after a preceding catch trial was significantly slower than that after any other SOA_{n-1} [$F(1,11) > 12.6, p < .01$, for all paired comparisons]. A deviation from this pattern was again observed for the shortest SOA_n, where responding after a preceding catch trial was faster than that after a preceding long SOA_{n-1} [$F(1,11) = 5.80, MS_e = 3{,}294.73, p < .05$].

Misses (i.e., failures to respond within 800 msec after target onset) occurred on 0.28% of the target trials and were not further analyzed. False alarms (i.e., responses on catch trials or responses earlier than 150 msec after target onset) occurred on 2.56% of all the trials. Paired comparisons indicated that significantly more false alarms occurred after a short SOA_{n-1} (5.20%) than after any of the other $SOAs_{n-1}$ [1.73%, 1.36%, and 1.96% for SOA_{n-1} of 500 msec, 900 msec, and catch, respectively; minimal $F(1,11) = 22.90, MS_e = 2.75, p < .01$]. Paired comparisons of the other three SOA_{n-1} conditions yielded no significant difference [maximal $F(1,11) = 1.15, MS_e = 1.92, p = .31$]. The response latency of the false alarms averaged 355 msec following the onset of the cue, with 92.17% of these responses occurring within an 150- to 500-msec interval.

Finally, to test the three-way interaction among cuing, SOA_n, and SOA_{n-1} on RT with maximal statistical

Table 1
Summary of the Analyses of Variance in Experiment 1 and Experiment 2

		Experiment 1		Experiment 2	
Source	*df*	MS_e	*F*	MS_e	*F*
Cuing	1,11	717.36	8.86*	1,439.23	21.11**
SOA on trial *n* (SOA_n)	2,22	1,785.62	10.77**	1,157.51	22.06**
SOA on trial *n*−1 (SOA_{n-1})	3,33	128.39	43.98**	196.85	13.94**
Cuing × SOA_n	2,22	154.31	77.65**	419.93	28.81**
Cuing × SOA_{n-1}	3,33	173.74	1.16	161.13	0.79
SOA_n × SOA_{n-1}	6,66	151.09	10.03**	200.16	11.32**
Cuing × SOA_n × SOA_{n-1}	6,66	141.21	1.14	117.99	0.98

*$p < .05$. **$p < .01$.

power, I combined the SOA conditions that Experiments 1 and 2 had in common (i.e., 100 msec, 500 msec, and catch trial). In this analysis, the three-way interaction was also far from significant [$F(2,46) < 1$].

Discussion

The results in Experiment 2 were very similar to those in Experiment 1. In fact, the only relevant difference was that for the shortest SOA, processing at the cued location was facilitated in Experiment 1, but not in Experiment 2. However, this difference between the experiments was numerically very small (10-msec facilitation in Experiment 1 vs. 5-msec facilitation in Experiment 2) and should not be of concern here. Another apparent difference was that IOR was significant for the middle SOA in Experiment 2, but not for the middle SOA in Experiment 1. However, this simply reflects the assumption that location-specific inhibition develops in real time after the onset of the cue, so that it has obtained its full-blown size after 500 msec, the middle SOA in Experiment 2, but not yet after 200 msec, the middle SOA in Experiment 1 (see, e.g., Klein, 2000; Posner & Cohen, 1984). Therefore, the findings of Experiments 1 and 2 are in close mutual agreement and are not dependent on a specific SOA range, which will allow me to focus next on their common features.

GENERAL DISCUSSION

In this study, two key findings with respect to IOR and nonspecific preparation were replicated. First, the significant interaction between cuing and SOA_n reflected the typical transition from facilitation to inhibition (i.e., IOR) of responding to a target at the cued, as compared with the uncued, location (see, e.g., Klein, 2000; Posner & Cohen, 1984). Second, the interaction between SOA_n and SOA_{n-1} reflected the typical phenomenon of sequential effects of SOA, which has been a key finding in studies of nonspecific preparation (e.g., Drazin, 1961; Los & van den Heuvel, 2001; Woodrow, 1914). The major question in this study was whether the mechanisms underlying IOR and nonspecific preparation operate independently. The finding that the interaction among cuing, SOA_n, and SOA_{n-1} was not significant strongly suggests that they do. In what follows, I first will focus on the justification and implications of inferring independent inhibitory mechanisms in space and time. Then I will discuss some more specific findings pertaining to these inhibitory mechanisms separately.

Inhibition in Space and Time

It will be noted that the inference of independent mechanisms in space and time relies on the acceptance of the null hypothesis. Even though this practice is problematic in general, there are several reasons why it is justified in the present case (cf. Frick, 1995). First, the nonsignificance of the three-way interaction was observed twice, in both Experiments 1 and 2. Second, in both experiments, the F value for the three-way interaction was close to 1, and, therefore, remote from significance. Third, the test of the three-way interaction yielded an F value below 1 even when power was maximized by combining the SOA levels that the experiments had in common. Fourth, statistical tests of interactions are generally more sensitive when the relevant factors consist of more than two levels, because this enables testing for the stability of a pattern, constituted by one or more factors, across the various levels of another factor (Sternberg, 1998). As Figures 3 and 4 show, a neat additive pattern of cuing across the four levels of SOA_{n-1} was apparent at each level of SOA_n. Fifth, in both experiments, both two-way interactions were highly significant, indicating that there was ample room for their mutual modification. In view of these arguments, the acceptance of the null hypothesis seems entirely justified.

The inference that IOR and sequential effects of SOA coexist without mutual interference is remarkable because both of these phenomena are generated during the cue–target interval and may rely on similar inhibitory mechanisms. In anticipation of the target, the mental system is told *not here* by the location of the cue, while it is independently told *not now* as a critical moment is bypassed. It should be noted, though, that, contrary to their simultaneous generation, the behavioral manifestation of these inhibitory effects are shifted by one trial. The inhibitory effect due to the spatial mechanism (i.e., IOR) is observed in the same trial as that in which it is generated, provided that there is enough time for its buildup (i.e., the SOA is longer than about 200 msec). By contrast, the inhibitory effect due to the temporal mechanism can become apparent only in the trial subsequent to its generation, because the inhibitory suppression under the temporal mechanism occurs only upon bypassing a critical moment. Note, though, that both inhibitory effects become manifest on the first possible occasion, which happens to be the current trial for the spatial effect and the subsequent trial for the temporal effect. So, the shift in the manifestation of inhibitory effects does not reflect any fundamental discrepancy between the two inhibitory mechanisms, and more important for present purposes, neither does it provide a clue about the observed additivity of inhibitory effects. The question remains as to why the inhibitory effect of the spatial mechanism, generated on the current trial, perfectly adds to the inhibitory effect of the temporal mechanism, generated on the preceding trial.

A plausible answer to this question is that the additive effect results from a modular organization of local inhibitory control processes involved in space and time. Several investigations have provided general support for inferring the involvement of separate modules from the additive effects of experimental factors (e.g., McClelland, 1979; Miller, 1993; Miller et al., 1995; Roberts & Sternberg, 1993; Sanders, 1998; Sternberg, 1998, 2001). At the same time, these investigations have seriously challenged the additional inference from additivity, once

deemed compelling (Sternberg, 1969), that the modules operate in a strictly serial fashion, as separate processing stages. On the basis of these insights, the present findings suggest a minimal modular, although not necessarily stage-like, architecture, in which the cuing and SOA_{n-1} factors selectively influence the duration of two separate modules, whereas SOA_n influences the duration of both modules. This solution implies that the inhibitory effect in space, as expressed by the interaction between SOA_n and cuing, originates from another module than the inhibitory effect in time, as expressed by the interaction between SOA_n and SOA_{n-1}. This raises the question of how to characterize the modules that are involved. I will consider two possibilities.

A first possibility starts from the consideration that space is inherently involved in any act of visual perception, whereas time is inherently involved in the planning, initiation, and maintenance of any motoric action. This view suggests that the inhibitory processes in space and time may be part of modules related to visual perception and motor action, respectively. There is converging evidence from a diversity of methods that (sequential) effects of SOA have a motoric locus (e.g., Coull, Frith, Büchel, & Nobre, 2000; Nobre, 2001; Rudell & Hu, 2001; Sanders, 1980). The status of IOR is less clear in this respect. A perceptual locus of this phenomenon seems to have been firmly established in studies in which it has been shown that IOR can be observed with measures of perceptual sensitivity (i.e., d'; Handy, Jha, & Mangun, 1999; but see Ivanoff & Klein, 2001) and with early occipital components of brain activity (McDonald, Ward, & Kiehl, 1999). However, other studies have supported a motoric locus, demonstrating the persistence of IOR under conditions of equal target visibility (Abrams & Dobkin, 1994) and the sensitivity of IOR to motoric manipulations (Ivanoff & Klein, 2001; Rafal, Calabresi, Brennan, & Sciolto, 1989). In the latter case, though, the inhibitory effect is probably concerned mainly with the oculomotor system (e.g., Kingstone & Pratt, 1999; Lepsien & Pollmann, 2002). Thus, to the extent that motor inhibition underlies IOR, it still seems to be related to acts of visual perception, of which eye movements are an integral part.

In a recent brain-imaging study, Coull and Nobre (1998; see also Nobre, 2001) directly compared the brain areas involved in spatial and temporal cuing. They used a task in which a cue could provide both temporal and spatial information about the impending target with 80% validity and observed that the brain areas involved in selective attention in space and time showed a good deal of specialization. Relative to the brain activation observed for neutral cues, spatial cues changed the activation in predominantly right-hemisphere brain areas typically included in perceptual and spatial processing, whereas temporal cues changed the activation in predominantly left-hemisphere areas typically involved in motor control. Of course, it is unclear whether these findings will generalize to the present study, in view of the differences in the experimental design (e.g., in the information value of the cue). At least, though, this study lends credibility to the proposed modular interpretation.

A second, although possibly related, characterization of the modules is suggested by evidence for modularity of attentional systems provided by Posner and colleagues. Thus, Posner and Petersen (1990) distinguished among an orienting system, involved in selecting sensory input, an alerting system, involved in the maintenance of an alert state, and an executive system, involved in resolving conflict among responses. There is evidence that these attentional systems are subserved by different neural networks and modulated by different neurotransmitters (see Posner & Fan, in press, for a review), supporting the claim of their modularity. Fan, McCandliss, Sommer, Raz, and Posner (2002) provided behavioral support for this view by using experimental factors that presumably taxed the different attentional networks differentially. Specifically, a visual target stimulus was either well or less well predictable in time (presumably taxing the alertness system), either well or less well predictable in space (presumably taxing the orienting system), and flanked by either congruent or incongruent stimuli (presumably taxing the executive network). The effects of these manipulations on RT were additive or nearly additive and showed very low mutual correlations across participants, suggesting that the attentional networks operate by and large independently.

When seen in the perspective of this general framework, the present findings suggest that IOR results from the operation of a local inhibitory mechanism in the orienting network, whereas sequential effects of SOA result from the operation of a local inhibitory mechanism in the alertness network. This is an interesting possibility, because it challenges the common notion that the orienting system is involved in selection, whereas the alertness system underlies general preparation. The present work suggests that the alertness system is no less selective than the orienting system, although with respect to a different dimension—time instead of space. The similarity between the embedded inhibitory mechanisms is a further illustration of a possible parallel between these systems that has hitherto gone unnoticed.

Facilitation in Space

Apart from inferences concerning the relationship between inhibition effects in space and time, the present study allows some comments on these phenomena when they are considered in isolation. As concerns the interaction between cuing and SOA_n, only a very small spatial facilitation effect on RT was found when the target appeared at the cued location after the shortest SOA, which turned out to be significant only in Experiment 1. However, this is not an uncommon finding (e.g., Danziger, Kingstone, & Snyder, 1998; Tassinari, Aglioti, Chelazzi, Peru, & Berlucchi, 1994). The magnitude of facilitation has been shown to depend on many factors apart from SOA (see, e.g., Pratt et al., 2001), for reasons that are often

not well understood. The present study suggests that the proportion of false alarms should also be considered as a contributing factor. False alarms occurred almost without exception briefly after the presentation of the cue. As a result, responses to targets occurring at the earliest critical moment are a mixture of target-based responses and false alarms that cannot be identified as such. By their nature, false alarms are not dependent on the cue–target relation, and so, to the degree to which they are present in the RT distribution for targets, they have the effect of reducing the difference between the cued and the uncued conditions, thereby leading to an underestimation of the cuing effect. Although the incidence of false alarms is low in most studies, including the present one, its potentially attenuating influence on cuing effects is noteworthy.

Inhibition in Time

The observed interaction between SOA_n and SOA_{n-1} replicated earlier findings, in that responding was slower to the degree to which the critical moment of target presentation was bypassed during the cue–target interval on the preceding trial (e.g., Los et al., 2001; Los & van den Heuvel, 2001). It should be noted that this empirical generalization is not fully consistent with the prediction derived from the conditioning model presented in the introduction. According to this model, the SOA_{n-1}–RT function should, for each SOA_n, feature a single step-like increase, occurring when SOA_{n-1} starts exceeding SOA_n, instead of the more gradual increase that was observed. However, as has been pointed out elsewhere (Los et al., 2001; Los & van den Heuvel, 2001), these are relatively minor deviations that can be dealt with in the spirit of the conditioning view by assuming that inhibition affects the state of conditioning more gradually over time.

By making possible an examination of the influence of sequential effects of catch trials, the present experiment provided a more pertinent test of the conditioning view. In a previous study, RT was shown to increase as a function of the number of preceding catch trials (Alegria, 1978). This led Alegria to suggest that the participant's general preparedness to respond decreases with the number of preceding catch trials. The conditioning view suggests a more comprehensive explanation by assuming that catch trials are essentially trials on which the cue–"target" interval extends beyond the latest critical moment. This view has considerable merit in that it provides a parsimonious explanation for the relatively sharp increase of RT for the longest SOA_n after a catch trial (right panels of Figures 3 and 4). This follows from the conditioning view because, for the longest SOA_n, a cost in RT should be observed only when the latest critical moment was bypassed during the preceding cue–target interval—that is, in the case of a catch trial. This is a relevant finding, because a large body of evidence indicates that for the latest critical moment, RT is relatively insensitive to whatever event occurred on the preceding trial.

On the other hand, the conditioning view has no clear explanation for the finding that for the shortest SOA_n, a preceding catch trial reverses the monotony of the SOA_{n-1}–RT function (left panels of Figures 3 and 4). In attempting to account for it, one may assume that the state of preparation corresponding to a bypassed critical moment gradually recovers from inhibition over time. If this recovery starts from the moment the participant drops his or her tense preparatory state (upon execution of the response on target trials, and upon realizing that no target will occur on catch trials), the present design probably allowed more time for recovery after catch trials than after target trials. To test the merits of this account, I examined RT for the shortest SOA_n (averaged across the levels of cuing) as a function of the duration of the preceding catch trial, which lasted 1,100, 1,200, or 1,500 msec (see also the Method section). This analysis failed to show any indication that RT decreased as a function of the duration of the preceding catch trial, arguing against the recovery account. Consequently, the reversal in the monotony of the SOA_{n-1}–RT function in Figures 3 and 4 suggests that it is not quite correct to conceive of a catch trial as a trial with a merely extended cue–"target" interval and that the all-or-none occurrence of the response itself should be considered as a factor of additional importance. This consideration may guide future developments of the conditioning theory of nonspecific preparation.

Conclusion

The present study has provided evidence for the independence of the inhibitory mechanisms operating on the spatial and temporal representations that give rise to IOR and sequential effects of SOA, respectively. This evidence suggests that these mechanisms operate as parts of different mental modules, perhaps involved in perception and action, respectively, and perhaps corresponding to the orienting and the alerting systems identified by Posner and colleagues (e.g., Posner & Fan, 2003). Still, this differential embedding should not obscure the functional resemblance between the inhibitory mechanisms, which suggests that the brain uses similar solutions in different domains of cognitive functioning. Future research may pursue the intriguing question of whether this resemblance is symptomatic of a more general equivalence of modules operating on spatial and temporal representations.

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